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DOWNY MILDEW OF THE BEET, CAUSED BY PERONOSPORA SCHACHTII FUCKEL[1]

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INTRODUCTION

The downy mildew, *Peronospora schachtii* Fuckel, on *Beta vulgaris* L. has been known in Europe since 1852 but was not observed in this country until 1911. Because of the supposed minor importance of this disease, no extensive studies of its prevalence and distribution had been made in America until 1929 when an epidemic of this disease destroyed over 40 per cent of the seed crop of the garden beet in central California.

Investigations showed that, although this disease has been known more than three-quarters of a century in Europe, comparatively little information was available regarding certain phases of the disease. Therefore, the pathogene was studied in regard to its life history, its host range, and the relation of environment to its attack.

THE DISEASE

Occurrence.—The downy mildew on *Beta vulgaris* was first described as *Peronospora schachtii* by Fuckel[(16)] in 1865 and was distributed as Fung. rhen. 1508. A slightly amplified description was

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published by the same author[17] in 1869. In 1872, Kühn[24, 25] presented a careful and accurate description of the macroscopic and microscopic symptoms of the disease, using the name *P. betae,* synonymously with *P. schachtii.* He stated that he first observed downy mildew of the beet in 1854 and that Schacht had observed the same disease sporadically in the years 1859 to 1861. The oospore stage of the causal fungus was not observed by Kühn[24] but he reported that by means of repeated experiments he had determined that *P. schachtii* overwintered as mycelium within the crown of the seed beets.

Prillieux[34] reported that downy mildew of the beet was first observed in France in 1852. He[33] first observed oospores of *Peronospora schachtii* in the leaves of diseased plants in 1882 and concluded that the fungus overwintered chiefly by means of these spores, rather than by hibernating mycelium as suggested by Kühn.[24]

Voglino[41] writing in 1899, agreed with Kühn as to the importance of hibernating mycelium and stated further that from such mycelium, condiophores and conidia may develop directly on the crown tissue of infected beets. He suggested that infected beets thrown on the refuse heap might provide inoculum for seedling beets during the following season. The production of conidiophores and conidia on the tissue of the beet crown has not been observed by other workers.

In England this disease was briefly studied in 1926 by Salmon and Ware,[38] who verified the presence of the mycelium within the beet crown, as shown by Kühn,[24] but were unable to find oospores. According to these authors, the first authentic report of the presence of *Peronospora schachtii* in England was in 1921, but Biffin[9] reported *P. schachtii* on mangels in England in 1913.

The first report of this disease in the United States was made in 1911 by Smith and Smith[40], who found infected sugar beets occurring to a limited extent in the coastal districts of California. Other reports from the same state have been made to the Federal Plant Disease Survey. Bensel[7] first recorded a severe outbreak of *Peronospora schachtii* on sugar beets in the Santa Clara Valley in 1927 and to a less extent in the Sacramento Valley the same year. He found from 1 to 66 per cent of the plants infected in the different fields and correlated the percentage of infection with the dates of planting and weather conditions. A similar outbreak, but of less intensity, occurred in 1925 in the same locality.

The only authentic report of *Peronospora schachtii* in other parts of the United States is that of a single infected plant of *Beta vulgaris* collected in New York on Long Island in 1917 and identified by H. S. Jackson (established through personal correspondence in 1929 with

Dr. Charles Chupp, Cornell University, Ithaca, New York). Gäumann[19] states that this fungus has also been collected in New Jersey and Minnesota. Communications from Dr. W. H. Martin of New Jersey and Dr. E. M. Freeman of Minnesota established the fact that there are no records of the collection of *Peronospora schachtii* in either state.

The geographic distribution of *Peronospora schachtii,* in so far as the writer has been able to determine, is as follows: Argentine, Belgium, Czecho-Slovakia, Denmark, Egypt, England, France, Germany, Italy, Japan, Palestine, Russia, Sweden, Switzerland, and United States (California and New York).

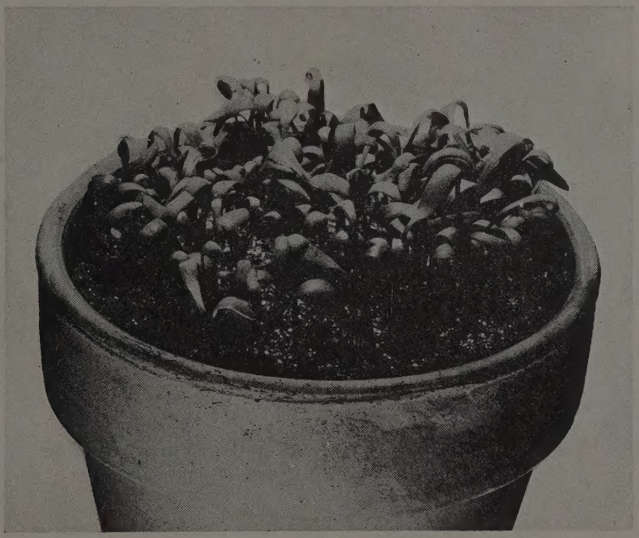

Fig. 1. Seedlings of *Beta vulgaris,* showing curling of the cotyledons caused by conidial infection of *Peronospora schachtii.*

Symptoms.—The disease exhibits several rather distinct stages of development, depending on the age and maturity of the host and on the ecological conditions. Symptoms of *Peronospora schachtii* infection on both young and old rosette leaves have been described by Kühn[24], Prillieux[33], and Voglino[41]. Salmon and Ware[38], and Hollrung,[22] described, in addition, the gross macroscopic effects of *P. schachtii* on the inflorescence. The symptoms on cotyledons of beet seedlings and on individual flower parts have not previously been reported.

When seedlings are infected while still in the cotyledon stage, the affected portions assume a color somewhat lighter than is normal for the variety, and the entire cotyledon usually curls downward (fig. 1). Under humid conditions a heavy coating of conidiophores and conidia appear on the upper as well as on the lower surface of the infected cotyledon and the young plants usually die from the effects of the disease. Cotyledon infection was readily produced by spraying with a conidial suspension under artificial conditions but has rarely been observed in field plantings.

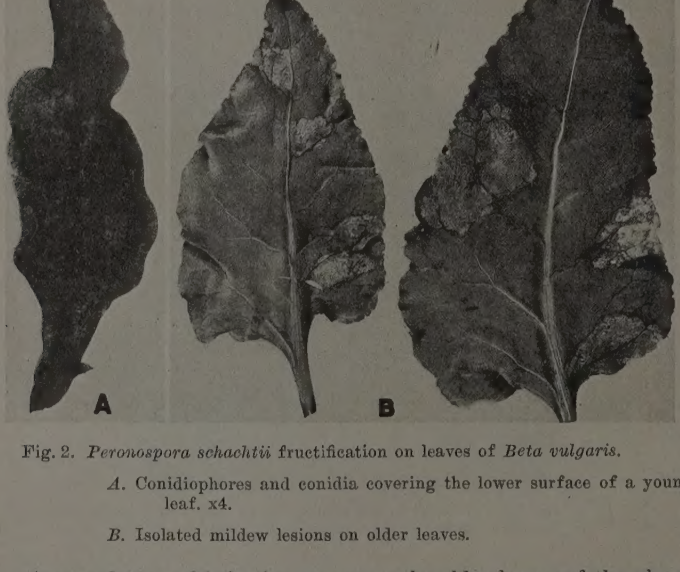

Fig. 2. *Peronospora schachtii* fructification on leaves of *Beta vulgaris*.

A. Conidiophores and conidia covering the lower surface of a young leaf. x4.

B. Isolated mildew lesions on older leaves.

A second type of infection occurs on the older leaves of the plants and presumably originates from air-borne conidia. The disease in this case manifests itself by isolated or sometimes coalescing spots of irregular shape, ranging in size from one to four centimeters in diameter (fig. 2*B*). These spots are differentiated from the healthy tissue by the lighter green color of the upper surface of the leaves and by the heavy coating of mildew fructification on the lower surface (fig. 2*A*). In this stage the effects of *Peronospora schachtii* on *Beta vulgaris*

bear a striking resemblance to those of *Peronospora effusa* (Grev.) Rab. on *Spinacia oleracea* L. During periods of extremely low humidity these spots on the beet leaves are sometimes surrounded by a narrow ring of pale red pigment. The symptoms described above are commonly observed in the fall-planted root beds (fields in which the seedling beets are maintained from the time of planting up to the age of three or four months) in the Sacramento Valley during the

Fig. 3. Effects of *Peronospora schachtii* on young leaves produced in the center of the rosette on severely diseased plants of *Beta vulgaris*.

A. Leaves showing infection along petiole and basal portion of the blade.

B. Distortion of inner rosette leaves invaded by the fungus.

C. Healthy leaf.

months of October and November and in the seed-beet fields (fields devoted to seed production) during March and April. In the coastal regions of California, where the temperature is usually somewhat lower and the humidity higher during the spring months, this type of infection can be observed as late as June.

Fig. 4. Effects of *Peronospora schachtii* on mature beets.

A and *B*. Severe infection on beet plants showing the curling, distortion, and stunting of the young rosette leaves.

C. Small shoot developing from a severely infected plant.

D. Floral shoots on healthy plant of the same age.

Late in the fall, and especially after a period of heavy rainfall, the symptoms of the disease most frequently described in literature develop. The young rosette leaves of the beet are attacked by the fungus and either the entire new leaf is covered by the mass of conidiophores or merely the basal portion of the blade and the petiole (fig. 3*A*). Microscopic studies show clearly that there is a continuous mycelial connection between leaves infected in this manner and the crown of the beet. Usually all new leaves produced subsequently show mildew infection in their entirety (fig. 4*B*). Such leaves are always small, thick, and often curled downward at the edges (fig. 3*B*). In most cases the color of infected leaves is light green but in some fields there seems to be a rather high correlation between the presence of *Peronospora schachtii* and red pigmentation upon the younger leaves. Conidiophores and conidia cover the lower surface of the leaves and also may appear on the upper surface under humid conditions. The presence of the fungus apparently acts as a stimulus to the production of new leaves, since on infected plants there are usually from two to twenty times as many leaves (fig. 4*A* and *B*) as on healthy plants growing under the same conditions (fig. 4*D*). In nearly all cases the mass of infected leaves is surrounded by a number of fully developed and apparently healthy rosette leaves (Fig. 4*A, B* and *C*). This condition is commonly found in seed-beet fields during the late winter and early spring months and is also the condition commonly observed in the market gardens and sugar beet fields. When the disease becomes extremely severe the affected leaves are killed and then are invaded by numerous saprophytic fungi and bacteria. The decay which results usually spreads down into the beet crown and finally the entire plant is destroyed. Many missing hills result from this process in infested fields. When the first floral shoots develop on infected plants usually all the leaves formed on them are stunted, curled, thicker than normal and, in many cases, covered with conidiophores and conidia. As the main axis elongates, however, the primary cauline leaves are often normal in appearance, although the axillary flower shoots are dwarfed and distorted by the fungus attack (fig. 5*C*). The sepals of infected flowers and the bracts at the base of the flowers become swollen and assume a light green color. During periods of cool, moist weather, conidiophores and conidia develop in large numbers on the sepals, bracts, leaves, and even on the axes of the secondary branches of the inflorescence (fig. 6*A*). On the smaller secondary branches many swellings or blisters, often covered with *P. schachtii* fructifications, are found. By micro-

scopic examination it was observed that the tissue underlying such areas is permeated by non-septate mycelium and haustoria characteristic of *P. schachtii*.

Fig. 5. Effect of *Peronospora schachtii* on the beet inflorescence.

A. Infected beet inflorescence in which leaf production has largely replaced flower production.

B. Compact floral shoots bearing infected flowers.

C. Infected flower shoots produced in the axils of apparently healthy cauline leaves.

D. Severely injured inflorescence branch bearing a few mature seed balls.

E. Portion of the inflorescence of a healthy beet plant.

Because the branches of the inflorescence fail to elongate, the flowers and seed balls (aggregates of two or more flowers fused at their bases) remain grouped together and the entire inflorescence

assumes the appearance of a compact cluster (fig. 5*B*) as compared with a normal paniculate spike (fig. 5*E*). Suppression of flower parts frequently occurs, accompanied by increased leaf production which results in the formation of a structure resembling a witches' broom (fig. 5*A*). The infected portions of the plant appear bleached and the

Fig. 6. *A. Peronospora schachtii* infection on the bracts and flowers of secondary inflorescence branches.

B. Healthy branch of approximately the same age.

hypertrophy of the individual flower parts is evident. Although in the case of severe infection most of the flowers are destroyed it is not uncommon to observe on the same shoots apparently healthy seed balls and sterile flower remnants covered with fructifications of *Peronospora schachtii* (fig. 5*D*). Observations on material from infected plants revealed many mature, viable seed balls which still carried the conidiophores of *P. schachtii* upon the dry, corky sepals (fig. 7*A*).

Economic Importance in California.—In central California losses caused by *Peronospora schachtii* occur in three types of beet plantings: market gardens, sugar-beet fields, and fields of garden beets grown for seed production. Garden beets are extensively grown in truck gardens along the coastal region of the San Francisco Bay

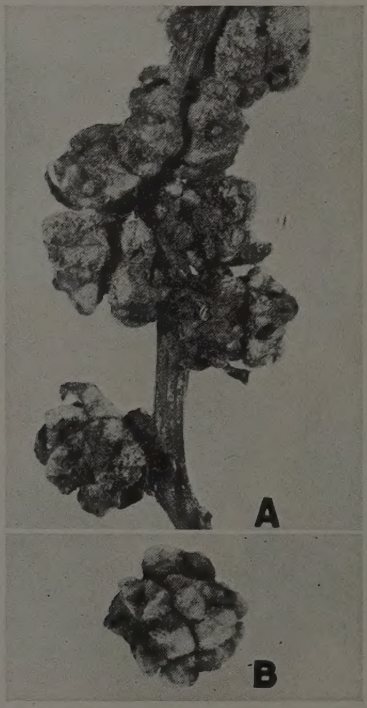

Fig. 7. Effect of *Peronospora schachtii* on seed balls of *Beta vulgaris.*

A. Mature seed balls covered with dry conidiophores of *Peronospora schachtii.* x4.

B. Apparently disease-free seed ball. x4.

district where, because of the high atmospheric moisture and the absence of high temperatures, *P. schachtii* occurs throughout the entire year. However, the greatest damage occurs in the months of January, February and March, during which period the amount of marketable beets is often reduced 25 to 50 per cent by the disease.

Peronospora schachtii has been observed on sugar beets since 1911 in the fields of the coastal region. Severe damage has not been reported except in the years of 1925 and 1927 when, according to Bensel,[7] 66 per cent of the plants in some fields were infected. Unfortunately there is no record of the effect of the mildew attack on the ultimate yield in the fields mentioned above. In recent years sugar beet production in the Santa Clara and Salinas valleys has been largely discontinued, partly because of the severity of curly top, a virus disease transmitted by the beet leafhopper (*Eutettix tenellus* Baker). At the present time large acreages of sugar beets are being produced in the Sacramento and upper San Joaquin valleys. A detailed survey of many fields in this area was made during the past two years and in no case was downy mildew found in destructive amounts. In the spring of 1929, approximately one per cent of the plants in one field were found to be infected, while in other fields, either no diseased plants or only a few were found. It was observed that in the latter case a single plant was often severely infected apparently without any spread of the disease to neighboring plants.

When sugar beets are planted in late November or December in the Salinas valley, sufficient growth occurs before the spring migration of the leafhoppers to enable the plants to partially withstand the effects of the curly-top disease. In one of the trial plantings near Salinas, California, seeded in November, 1929, 73 per cent of the plants were found to be infected with *Peronospora schachtii*. Determinations made in the above-mentioned field indicate that the tonnage was reduced 17.3 per cent, the sugar percentage was lowered 1.46, the percentage of purity 4.96, and the total sugar yield was reduced 29.3 per cent, by the downy mildew epidemic. Since winter planting is becoming a commercial practice in the Salinas Valley, it is probable that downy mildew will become a more important factor in sugar beet production than is now the case.

Although no survey of sugar beet fields in southern California has been made by the writer, the disease is known to occur there, since specimens infected with *Peronospora schachtii* have been sent in for identification.

By far the greatest losses caused by this disease in central California occur in the fields devoted to the production of garden beet seed (fig. 8). In 1929 an estimate of the reduction in yield caused by downy mildew in the 600 acres devoted to beet-seed production in the lower Sacramento Valley, showed that 23 per cent of the acreage was abandoned as a total loss, 17 per cent suffered a 75–90 per cent

loss, 18 per cent a 25 per cent loss, and 42 per cent a loss of less than 5 per cent. A conservative estimate of the total reduction in yield of the 600 acres was 43 per cent. The actual loss caused by the disease averaged $100 per acre or a total of $60,000. During the season of 1930, the disease was present in most of the seed fields but the damage was less severe than in the previous year. The reduction in yield for the entire district was estimated at 25 per cent.

Fig. 8. *A*. Detroit Dark Red beet seed field in which 95 per cent of the plants were infected with downy mildew. Clarksburg, California, May 2, 1929. Yield: 214 pounds of seed per acre.

B. Detroit Dark Red beet seed field in which only 3 per cent of plants showed downy mildew injury. Clarksburg, California, May 24, 1929. Yield: 2,200 pounds of seed per acre.

Under central California conditions the methods of beet-seed production have been modified so that the beet plant, a true biennial, completes its life cycle within a period of ten to twelve months. During August or September the seed is planted in rows 15 inches apart in root beds where, forced by frequent irrigation, the plants produce stecklings (small beet roots used for commercial seed production) from one-half to two inches in diameter before the cool weather of November and December occurs. After the fall rains permit the preparation of fields, the beet plants are transplanted direct from the root beds to the seed fields where they are spaced from 36 to 40 inches apart. The transplanting operation is usually completed in December or January. During the winter months growth is confined chiefly to root development but in March the rosette leaves develop rapidly, followed in April and May by the formation of the floral axis. Usually the mature seed balls are harvested during July in the interior valleys and during August in the coastal regions.

The losses from *Peronospora schachtii* on garden beets grown for seed production are due: first, to injury on beet plants in the root beds before transplanting to the seed fields; second, to death of plants in the seed fields; and third, to reduction in yield of the infected plants which survive.

Peronospora schachtii infection on beet seedlings in the root beds, while it does not severely injure the individual plants, is important in relation to the development of the disease in the seed fields. Seedlings may be weakened by the severe attack of *P. schachtii* on the leaves in the early stages of development, but after the fungus becomes established in the young rosette leaves the effect on the plant is much more marked. Such plants are practically worthless for transplanting and are a dangerous source of infection to nearby healthy plants. Observations have shown that downy mildew in the seed fields can, in nearly all cases, be traced directly to root bed infection. For this reason entire root beds are sometimes discarded when the percentage of infected plants is high, rather than risk transferring diseased plants to the seed fields.

During the spring months, *Peronospora schachtii* produces its most severe injury on the seed beets. Individual plants are so severely attacked that the rosette leaves of the plants become a stunted and distorted mass (fig. 4*B*). The death of certain of the leaves apparently provides an entrance for numerous saprophytic fungi and bacteria, which probably assist in completing the destruction of the plants. The reduction in stand in many cases constitutes the greatest single loss

from this disease. Infected plants which survive until warm weather occurs usually produce poorly developed inflorescences, and the amount of salable seed is always less than that produced by disease-free plants.

In order to secure information on the dissemination of *Peronospora schachtii*, a study of the disease as it appeared in root beds was undertaken. Ten root bed plantings were kept under observation to determine, as far as possible, how many original infections occurred, and the rate of spread of the disease in the field.

During the fall of 1929 the rainfall in the Sacramento Valley was unusually light, less than 0.15 inches having fallen before December 8. Conditions were relatively unfavorable, consequently, for the development and spread of the mildew, making it possible in the case of one isolated field to trace the development of the epidemic from its origin. This field of 1¾ acres was planted to the variety Detroit Dark Red on August 19, 1929, with seed of unknown origin. Observations made on August 28 and September 20 failed to reveal any infected plants but on October 10 one plant was found with severe *Peronospora schachtii* infection on several of the older leaves. The conidial mass on some lesions was very dark and beginning to dry up, indicating that infection had occurred at least two weeks before the date of observation. Early stages of infection were found on about 25 per cent of the plants within a radius of eight feet from the heavily infected plant mentioned above (fig. 9*A*). A careful survey of the remainder of the field, however, failed to reveal any other diseased plants. In order to determine the rapidity of spread, observations were made at intervals of one week throughout October and November. During this period the only moisture available for conidial germination on the leaves was dew, which however, was abundant throughout most of the period because of the surface irrigation practiced.

By October 18 the symptoms of disease were observed to have spread a distance of 90 feet, or entirely across the narrow field, consisting of five plant beds (fig. 9*A*). By October 25 the downy mildew had spread over one entire end of the planting and a small infected area appeared in the opposite end of the field (fig. 9*B*). As in the other case, one heavily infected plant was surrounded by others bearing evidence of lighter and more recent infection. Because of the distance and the absence of the disease between the two areas, the second is believed to be another case of original infection. It is possible, however, that it may have resulted from wind-blown conidial infection from the other end of the field, some 450 feet distant.

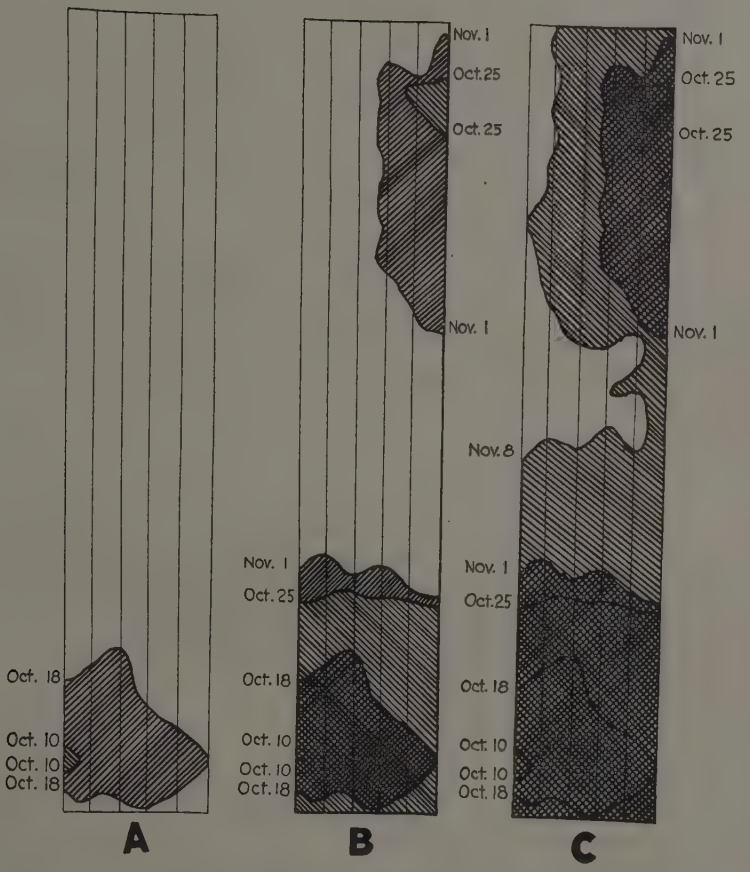

Fig. 9. Successive development of an epidemic of *Peronospora schachtii*.

A. Beet root bed showing areas found to be infested on October 10 and 18, 1929.

B. Same root bed showing areas invaded up to November 1, 1929.

C. Same root bed showing the spread of the disease from October 10 to November 8, 1929.

By November 1 both areas were somewhat enlarged but maintained their identity. One week later the two areas had coalesced along one side of the field and only a small area in the center and along one side remained free from infection (fig. 9*C*). After this date heavy frosts frequently occurred, and either this fact or the stage of development of the beet leaves seemed to be unfavorable for the spread of the disease. No new infested areas were observed on November 15 and the infection on many of the plants appeared to be less severe than formerly. Up to this time all the downy mildew lesions, with a few exceptions, occurred as isolated areas on the outer, well-developed leaves. Soon after the advent of the late fall rains, heavy infection of the inner rosette leaves could be observed, while the first type of infection practically disappeared. The greatest number of plants bearing the second type of infection were found in the areas in which the disease had appeared at an early date.

These observations indicate that infection may appear on seedling beets in the field within a month after their emergence. In the case of the field studied, all of the infections could be accounted for by wind-blown conidia except two, which were considered to be original infections. This fact explains how an epidemic covering an entire field may result from a few cases of successful seed transmission or oospore infection. Because of the rapidity of the spread even under relatively unfavorable conditions, it is evident that any attempt to control downy mildew in beet root beds by spraying or eradication must take into consideration the fact that the limits of infection at any given time extend considerably beyond the area showing visible symptoms of the disease.

HOST RANGE

Peronospora schachtii has been reported on the following hosts: sugar beets, garden beets, mangels (*Beta vulgaris*), and wild beets (*B. maritima* L.). In addition Ravn[35] reports *Peronospora schachtii* on the "Bladbede" while a popular article from Egypt[2] refers to the presence of spinach mildew (*P. effusa*) on Swiss chard. In May 1929, a downy mildew was observed on Swiss chard (*B. vulgaris* var. *cicla* L.) in the experimental plot at Davis, California. The fungus resembled *Peronospora schachtii* morphologically and readily infected garden beet seedlings, while *P. schachtii* conidia from garden beets infected Swiss chard seedlings under similar conditions. Natural infection of Swiss chard was later found on two occasions in the market garden section south of San Francisco. In repeated trials the

writer failed to produce infection on seedlings of Swiss chard with conidia of *P. effusa* from *Spinacia oleracea*, while on the other hand, Swiss chard seedlings were readily infected by conidia of *Peronospora schachtii*. From this evidence it appears that Swiss chard is susceptible to *P. schachtii* but not to *P. effusa* from *Spinacia oleracea* in California.

Since little information concerning the host range of *Peronospora schachtii* was available it seemed desirable to test the relative susceptibility of varieties of garden beets, sugar beets, and mangels, as well as to test the susceptibility of species of *Beta, Spinacia,* and *Chenopodium.*

All the plants to be tested were inoculated while in an early seedling stage, by spraying them with a conidial suspension. The seedlings were then incubated at 8° C for 48 hours in a saturated atmosphere, a temperature shown later in this paper to be favorable for infection. At the end of this time the seedlings were transferred to moist chambers on a greenhouse bench. On susceptible plants, sporulation occurred five or six days after inoculation and after eight days a record was taken of the percentage of plants showing the presence of conidiophores and conidia. It was found that the age of the seedlings greatly influenced the percentage of infection, the older plants being less susceptible. For that reason all comparisons are based on results obtained with young seedlings and these are divided into two age classes, the first from 11 to 20 and the second from 21 to 30 days after planting.

The investigations reported in table 1 were conducted between November 1929 and March 1930. The results with each variety represent the compilation from two or more trials. Eight varieties were inoculated at one time and seedlings of the variety Detroit Dark Red were included with each set so that a comparison of the separate trials could be made. Since over 90 per cent of the 11 to 20-day-old seedlings of the variety Detroit Dark Red, and over 80 per cent of those from 21 to 30 days of age were infected in every case, the results of all trials were brought together in one table.

Twelve commercial varieties of garden beets were tested and all were found to be susceptible in the seedling stage. There were, however, some differences and from these trials it appeared that the varieties Crosby's Egyptian and Detroit Dark Red were the most susceptible, while the varieties Long Smooth Blood and Crimson Globe were relatively resistant. It seems probable from the few trials that have been conducted that older plants of certain varieties may be markedly resistant to the attack of this fungus.

All of the nine mangel varieties tested showed some infection, but the variety Long Red seemed to be the most resistant. However, the number of seedlings of this variety was too few to permit of definite conclusions. Two sugar beet varieties, Kleinwanzleben and an unlabeled commercial variety were not as susceptible as the majority of garden beet varieties, but were readily infected in the seedling stage.

TABLE 1

COMPARATIVE SUSCEPTIBILITY OF CULTIVATED VARIETIES OF BETA VULGARIS TO PERONOSPORA SCHACHTII

Common name and variety		Inoculated 11 to 20 days after planting			Inoculated 21 to 30 days after planting		
		Total plants	Number infected	Per cent infected	Total plants	Number infected	Per cent infected
Garden beets	Crimson Globe	75	52	69	103	64	62
	Crosby's Egyptian	89	88	99	136	131	97
	Detroit Dark Red	515	499	97	307	270	88
	Early Blood Turnip	101	97	96	92	56	61
	Early Eclipse	109	105	96	49	37	76
	Early Model	97	95	98	50	35	70
	Early Wonder	127	91	72	....	....	....
	Extra Early Egyptian	170	167	98	83	71	86
	Fireball	107	104	97	99	85	86
	Half Long Blood	95	87	92	172	101	59
	Half Long Special	183	134	73	182	147	81
	Long Smooth Blood	229	119	52	159	64	40
Mangels	Danish Slugstrup	132	117	89	169	125	74
	Giant Half Sugar Rose	225	191	85	109	71	65
	Golden Giant Intermediate	225	164	73	135	82	61
	Golden Tankard	86	80	93	83	50	60
	Long Red Mangel	22	5	23	27	3	11
	Mammoth Long Red	183	159	87	231	72	31
	Red Eckendorf	115	102	89	160	130	81
	White French Sugar	90	80	89	144	94	65
	White Sugar Rose Top	84	75	89	125	112	90
Sugar beets	Commercial sugar beets (variety unknown)	49	38	78	83	37	45
	Beta vulgaris var. *plantaginifolia*	200	86	43	85	3	4
	Kleinwanzleben	128	76	59	220	121	55
Swiss chard	Giant Lucullus	171	55	32	148	27	18
	White Silver	223	156	70	118	58	49

A sugar beet selection designated as *Beta vulgaris* var. *plantaginifolia* Zalenski was found to be resistant, especially when exposed to infection after the first true leaves had developed. It is interesting to note in passing that this selection of sugar beets was reported by

Shevchenko[39] as highly resistant to infection with *Cercospora beticola* Sacc. Both varieties of Swiss chard were susceptible to downy mildew, but Giant Lucullus showed more resistance.

Ten *Beta* species were inoculated by spraying them with conidia from garden beets and all became infected (table 2). Of those tested, *Beta bourgaei* Coss. and *B. patula* Soland. were apparently the most resistant. Samples of *B. maritima* obtained both from Europe and California were exposed to infection and over 70 per cent of the seedlings were infected in each case. *Chenopodium album* L. and *C. murale* L. were repeatedly sprayed with conidial suspensions but no infection occurred.

TABLE 2

COMPARATIVE SUSCEPTIBILITY TO INFECTION WITH PERONOSPORA SCHACHTII OF WILD SPECIES OF BETA AND CHENOPODIUM

Genus and species		Inoculated 11 to 20 days after planting			Inoculated 11 to 20 days after planting		
		Total plants	Number infected	Per cent infected	Total plants	Number infected	Per cent infected
*Beta**	*bourgaei*	26	7	27	....	....	....
	bourgaei x B. procumbens	49	41	84	49	36	74
	macrocarpa	53	52	98	34	29	85
	maritima	270	211	78	209	146	70
	patellaris	....	....	....	141	75	53
	patula	21	8	38	.....	.....	.....
	procumbens	53	51	96	.....	.....	.....
	scutellaris	28	23	82	.....	.....	.....
	vulgaris var. *abyssinica*	56	49	88	34	23	68
Chenopodium	*album*	....	....	....	32	0	0
	murale	36	0	0	26	0	0

* The seed of the *Beta* species was furnished through the courtesy of Vilmorin-Andrieux and Company, Paris, France. Although originally collected in various parts of Europe the species have been growing in close proximity to each other for several years with every opportunity for cross pollination and therefore cannot be considered pure.

All of the species and varieties of the genus *Beta* which were tested in these trials were found to be susceptible in the seedling stage although there was, in some cases, a marked difference in their response.

Morphologically *Peronospora schachtii* and *P. effusa* resemble each other closely and both infect members of the Chenopodiaceae. In an attempt to compare the pathogenicity of the two fungi, three hosts of *P. schachtii,* Detroit Dark Red beets, White Silver chard, and Giant Lucullus chard; and two hosts of *P. effusa,* Prickly Winter and Long Standing spinach, were sprayed with conidia from *Beta vulgaris* and *Spinacia oleracea.* The results are given in table 3.

In table 3 the percentage of infection in each case was secured by trials, in duplicate, conducted in March 1929. Conidia from *Beta vulgaris* readily infected the variety of garden beets, Detroit Dark Red, and the Swiss chard varieties, White Silver and Giant Lucullus, but produced no infection upon two varieties of Spinach, Prickly Winter and Long Standing. On the other hand, conidia from *Spinacia oleracea* produced no infection on the same varieties of garden beets and Swiss chard while readily infecting the two varieties of spinach. The results show that, as far as the varieties tested are concerned, the host ranges of the two species of *Peronospora* are distinct.

TABLE 3

COMPARISON OF THE HOST RANGE OF PERONOSPORA SCHACHTII AND PERONOSPORA EFFUSA ON VARIETIES OF BETA VULGARIS AND SPINACIA OLERACEA

Host	Variety	Conidia of *P. schachtii* from *Beta vulgaris*			Conidia of *P. effusa* from *Spinacia oleracea*		
		Total plants	Number infected	Per cent infected	Total plants	Number infected	Per cent infected
Beta vulgaris	Detroit Dark Red	150	147	98	135	0	0
Beta vulgaris var. *cicla*	White Silver	122	87	71	147	0	0
B. vulgaris var. *cicla*	Giant Lucullus	108	39	36	73	0	0
Spinacia oleracea	Prickly Winter	130	0	0	117	115	98
S. oleracea	Long Standing	79	0	0	65	58	89

CAUSAL ORGANISM

A description of *Peronospora schachtii* has been given by Fischer,[15] Berlese,[8] and Riehm[36] and will not be repeated here. In the course of these studies certain points have been found which have not been previously presented or which differ from previous descriptions. The coarse, non-septate mycelium can be satisfactorily differentiated from the host tissue in free-hand sections by staining for a few minutes with cotton blue in lacto-phenol. The diameter of the mycelium has been found to vary from 4.4μ to 11.0μ, the average being 6.7μ.

According to Prillieux[34] and Voglino[41], one to three condidiophores emerge from a single stoma. A microscopic observation on young diseased cotyledons has shown in some cases as many as ten conidiophores protruding from a single stoma. From the enlarged

conidiophore base, measuring about 11μ in diameter, the trunk gradually tapers until at the base of the branches the diameter averages approximately 5.7μ. Observations on 200 conidiophores produced in a laboratory moist chamber at 13–15° C have shown that the number of sterigmata on a single conidiophore varied from 5 to 43, the mean being 15 $\pm$ 4.4. The total length of the conidiophores varied from 177μ to 653μ, the mean length being 356 $\pm$ 41.4μ. The trunk of the conidiophore occupied from two-thirds to three-fourths of the total length, while the branched portions constituted the remainder. These measurements are somewhat larger than those observed by Voglino[41] and Berlese,[8] who stated that the conidiophores were from 250μ to 350μ in length.

Gäumann[19] has shown that the size of conidia in species of *Peronospora* may be greatly influenced by the temperature and humidity during the period of formation and by the stage of maturity of the spore. These results emphasize the importance of making spore measurements under standard conditions which may be duplicated by other workers. Peters[32] apparently made his measurements on fresh conidia while Gäumann used conidia from specimens in exsiccati. In the present studies, measurements have been made on both types of material in order to be able to draw direct comparisons. In the first trial, observations were made on conidia produced on leaves of *Beta vulgaris* in a moist chamber containing a saturated atmosphere at a temperature of 13–15° C. Under these conditions abundant conidia, uniform in size, shape, and general appearance, developed. Germination and infection experiments showed that conidia produced in this manner are functionally mature. Measurements were made with a 4 mm objective and filar micrometer on fresh conidia mounted in tap water. All conidia were disregarded which were obviously immature or which did not present their greater axis at right angles to the line of vision.

In the second trial, conidia which had been dried on a leaf of *Beta vulgaris* in the laboratory for a period of 15 days were used. Following the methods of Gäumann[19], the dried spores were placed in concentrated lactic acid to restore turgidity. The results obtained by measuring 200 fresh and 100 dried conidia are presented in table 4 in comparison with the measurements by Fischer,[15] Voglino,[41] Berlese,[8] Peters,[32] Gäumann,[19] and Riehm.[36] The similarity of the results obtained with fresh and dried spores indicates that the size of conidia is not greatly influenced by the methods used in preparing them for measurement. The writer's measurements correspond

closely to the results of Peters[32] and Fischer,[15] and although the average length of conidia is somewhat less than that recorded by Gäumann[19] and Riehm[36] the differences probably are not significant.

Oospores of *Peronospora schachtii* were first observed in 1882 by Prillieux[33] who reported that they were abundant in the leaves of infected plants. Kühn[24] found no oospores in Germany and at a much later date Peters[32] reported that oospores rarely, if ever, formed in that country.

TABLE 4

MEASUREMENTS OF CONIDIA OF PERONOSPORA SCHACHTII COMPARED WITH MEASUREMENTS OF PREVIOUS WORKERS

Authority	Length in microns		Width in microns	
	Range	Mean	Range	Mean
Fischer[15]	21–26	25	16–21	20
Voglino[41]	20–24	....	15–18	....
Berlese[8]	22–27	....	17–20	....
Peters[32]	.....	24	.....	20
Gäumann[19]	.....	27	.....	21
Riehm[36]	.....	27	.....	21
Present studies, fresh conidia	20 3–28.1	24.0±.96	17.5–24.3	20.2±.81
Present studies, dried conidia	21.9–28.5	24.5±.82	17.5–23.5	19.9± 76

In the San Juan District of San Benito County, California, near the coast, many oospores were found in the infected leaves of beet plants after dessication had occurred. In the Sacramento Valley, however, during the latter part of the growing season when hot, dry weather prevailed, oospores were found only occasionally. The formation of the sexual spore in these cases apparently occurred in connection with the maturity or death of the host tissues. Oospore formation, however, can take place in young, actively growing leaves. Under artificial conditions oospores have been produced in cotyledons and young leaves within 30 days after they were sprayed with a conidial suspension (fig. 10*A*). In such cases the same leaf areas seldom produced both conidiophores and oospores. There is some evidence that low atmospheric moisture inhibits conidiophore production in young seedlings and stimulates the formation of sexual spores. In one young leaf oospores and oogonia were found at the rate of 29,000 per square centimeter of leaf area.

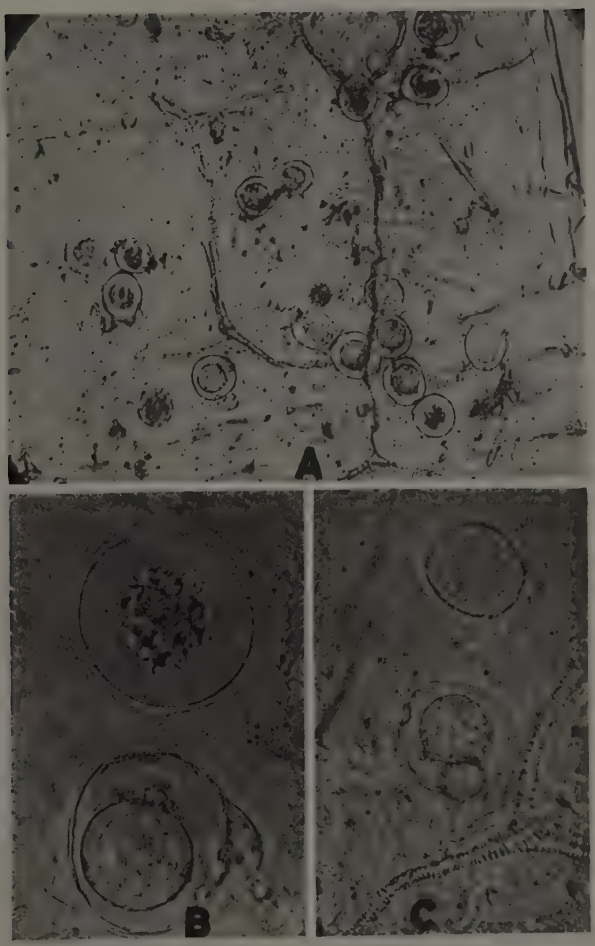

Fig. 10. *A*. Oogonia of *Peronospora schachtii* in beet leaf infected under artificial conditions.

B. Two stages in the development of the oospore; the upper showing the rounding-up of the contents in the center of the oogonium and the lower showing the formation of the oospore wall. An old antheridium remains attached to the oogonium in the latter. x565.

C. Oogonium with antheridium closely appressed to its surface is shown in the center. x330.

The young oogonium of *Peronospora schachtii* appears as a globular, many-nucleated structure surrounded by a thin membrane, the oogonial wall. A single paragynous antheridium usually appresses itself to the periphery of the oogonium (fig. 10*B* and *C*), and the contents of the oogonium round up in the center and become delimited from the periplasm by a definite membrane which thickens to form the oospore wall (fig. 10*B*). Later the periplasm is deposited on the oospore wall as a smooth but irregular epispore, the thin oogonial wall collapsing with the maturity of the oospore. Within the oospore, reserve granules in different states of development have been observed. By measuring 50 apparently mature oospores, it was found that the oospore diameter ranged from 26.6μ to 35.6μ with a mean diameter of 30.8 $\pm$ 1.54μ. It was also found that the oospore wall was from 1.2μ to 3.4μ in thickness with a mean of 2.2 $\pm$ .21μ.

Germination of oospores of comparatively few species of the Peronosporales has been reported. De Bary[6] has shown that the oospores of *Peronospora valerianellae* Fuckel and *Pythium de Baryanum* Hesse germinate by a germ tube while those of *P. proliferum* de Bary and *Phytophthora omnivora* de Bary germinate by a tube ending in a terminal sporangium. De Bary[5] has also shown that oospores of *Cystopus candidus* (Pers.) Lev. germinate by zoospores. The literature on the germination of oospores of *Plasmopara viticola* (Berk. and Curt.) Berl. and de Ton. has recently been compiled by Arens[3]. The germination of oospores of *Pseudoperonospora humuli* (Miyabe and Takah.) Wils. by the production of sporangia which liberate zoospores was shown by Arens.[4] Hiura[20] and Evans and Harrar[14] have shown that cospores of *Sclerospora graminicola* (Sacc.) Schroet. germinate by a germ tube. Peglion[31] on the other hand has shown that the oospores of *S. macrospora* Sacc. produce macroconidia upon short germ tubes. Oospores of *Peronospora spinaciae* (Grev.) Laubert (*P. effusa*) were reported by Eriksson[12] to germinate by a germ tube without passing through a resting period. The only record of the germination of oospores of *P. schachtii* is that reported by Voglino[41] who stated that oospores placed on a dry leaf of *Beta vulgaris,* germinated by germ tubes, while those placed in drops of water on the leaves germinated by means of zoospores.

During these studies numerous attempts to germinate the oospores of *Peronospora schachtii* have been made without success. This may, in part, be attributed to immaturity of the oospores or to unfavorable conditions of storage. Two methods have been employed in the germination trials. In one, the finely pulverized leaf tissue containing

the oospores, was dusted onto the surface of tap water in watch glasses. The floating cultures were stored at different temperatures between 2° C and 30° C and observations were made at the end of 24 and 48 hours. In the second method, the oospore material was dusted on a piece of filter paper, which was supported on a wire frame above a free water surface and enclosed in a small moist chamber. At the end of 24 and 48 hours, portions of the oospore material were carefully scraped from the filter paper and examined for signs of germination. None was observed in any of the trials.

Conidial Germination.—In early germination trials, conidia were secured from diseased leaves collected out-of-doors early in the morning. In some cases excellent germination was secured by using the conidia removed from such leaves, while at other times not over 50 per cent germination was obtained at the most favorable temperatures. In order to secure more uniform results, heavily infected leaves, collected in the late afternoon, were washed under a strong stream of water while the surface of the leaves was rubbed with the fingers to remove the conidiophores and conidia. The infected leaves were then placed in a moist chamber and held at 13–15° C for 18 to 24 hours, during which time an abundance of fresh conidia developed. A few drops of tap water were placed on the sporulating surface, and repeatedly drawn up into and expelled from a medicine dropper. When the water drops became turbid, due to the presence of large numbers of conidia, the spore suspension was used immediately for the preparation of hanging drop cultures. By using this method uniformly viable conidia, free from foreign material, were secured.

All trials on the relation of temperature to percentage of germination, rate of germination, and rate of germ-tube growth, were made by means of spore suspensions in hanging drop cultures. At first both Van Tieghem cells and hollow-ground slides were used, but after a comparison had shown that there was little or no difference in the percentage of germination by the two methods, only the latter was used.

A simplified technique for making hanging drop cultures with hollow-ground slides was devised. As far as is known, this method is not in use in other laboratories. All of the hollow-ground slides to be used in a series of trials were placed on a laboratory table with the concave surface upward. A glass ring, of approximately the same diameter as the concave area of the slide, was dipped into melted vaseline and quickly touched to each slide in such a manner that a circle of vaseline was formed around the concave area. The required

number of cover slips (25 mm square) were then arranged on the table and a small drop of the spore suspension placed in the center of each. A slide prepared as stated above was then inverted over each cover slip and gently pressed down until the vaseline established contact between the slide and cover slip, forming an air pocket above the drop. The slide and cover slip were then inverted by a quick, continuous motion, without disturbing the drop and the culture was ready for observation or incubation.

Temperature Relation.—For these studies the hanging drop cultures were incubated for 18 to 24 hours in thermostatically controlled chambers, the temperature of which varied less than 1° C. The percentage of germination at each temperature was obtained by counting the spores in four or five fields on each of two or more slides incubated at that temperature. Between 100 and 300 spores were counted on each slide, depending upon the uniformity of germination in the drop.

TABLE 5

Effect of Temperature* on the Percentage Germination of Conidia of Peronospora Schachtii Collected Under Field Conditions

Trial	½°	2°	4°	6°–7°	8°	10°	13°	15°–17°	19°	21°	23°–25°	27°–28°	30°
1	....	....	92	82	65	....	11	6	....	6	....	1	....
2	14	21	37	44	38	19	7	6	....	5	....	....	....
3	1	19	44	34	39	27	30	....	6	4	....	....	....
4	5	22	29	45	47	31	24	....	9	8	....	....	....
5	23	82	88	79	....	55	....	3	1	2	1	....	0
6	40	69	83	80	....	75	....	24	10	10	7	....	0
7	30	51	51	62	....	52	....	21	....	6	5	....	0
Maximum per cent germination	40	82	92	82	65	75	30	24	10	10	7	1	0

* Temperature in degrees Centigrade.

The conidia used in all of the trials shown in table 5 were collected out-of-doors in early morning and their lack of uniform maturity accounts for the relatively low percentage of germination at all temperatures in trials 2, 3, 4 and 7. The trials in table 5 were conducted between April 13 and May 4, 1929. The results are not sufficiently uniform to show definitely the optimum temperature, but these studies indicate that 4° C is the most favorable for the germination of conidia of *Peronospora schachtii.* Because of the wide differences in germination at the same temperature, the highest percentage of germination at each temperature represented most accurately the capacity of the conidia for germination.

TABLE 6

EFFECT OF TEMPERATURE* ON THE PERCENTAGE GERMINATION OF CONIDIA OF PERONOSPORA SCHACHTII PRODUCED IN A MOISTURE CHAMBER

Trial	½°	2°	4°	6°–7°	8°	10°	13°	15°–17°	21°	23°–25°	27–°28°	30°
1	33	93	98	97	....	26	....	3	1	2	....	1
2	....	96	98	98	97	34	....	2	1	1	1	0
3	30	89	97	99	99	61	....	4	1	4	1	....
4	66	74	82	....	87	86	9	....	4	....	1	0
5	4	67	94	....	16	2	1	....	1	6	....	0
6	34	86	96	....	54	32	5	....	1	3	....	1
Total number conidia counted	1099	1873	2085	1020	1600	1943	956	942	2102	2298	1259	2130
Total number germinated	295	1602	1974	999	1147	692	45	25	26	71	11	3
Per cent germination	26.9	85.5	94.5	98.0	71.5	35.6	4.7	2.7	1.2	3.1	0.9	0.1

* Temperature in degrees Centigrade.

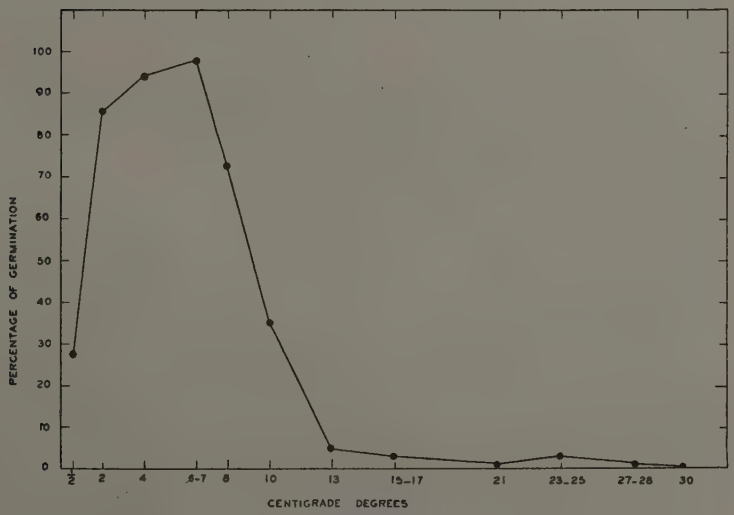

Fig. 11. Relation of temperature to the germination of *Peronospora schachtii* conidia.

The conidia used for the trials reported in table 6, instead of being collected out-of-doors, were produced in a laboratory moist chamber on infected leaves from which the previous conidial coating had been removed by running water. The total number of conidia counted, the total number which germinated, and the percentage of germination for all six of the trials are shown in the summary at the bottom of table 6. The percentage of conidial germination obtained at each temperature from ½° to 30° C is shown graphically in figure 11.

It is evident from the results presented in tables 5 and 6 that, under the conditions of these experiments, the optimum temperature for the conidial germination of *Peronospora schachtii* is from 4° to 7° C. The maximum temperature is approximately 30° C, since only a few trials showed any germination at that temperature. From 4 to 66 per cent germination was obtained at ½° C; therefore, the minimum temperature for conidial germination is apparently near the freezing point.

Although the summary of all the trials shows a higher percentage of germination at 6–7° C than at 4° C, a comparison of the results in trials where the same conidial suspensions were incubated at each of the two temperatures, shows no significant differences. Less than ten per cent germination was obtained in the trials conducted at temperatures above 10° C.

Because of the extreme tolerance of the conidia for low temperature, it was decided to study the effect of freezing on subsequent germination. Duplicate series of hanging drop cultures were prepared and one, the control series, was immediately incubated at temperatures ranging from ½° to 30° C, while the other was frozen at –12° C for 24 hours before being distributed over a similar range of temperatures. The experiment was repeated and the results of both trials showed that conidia were not killed by freezing at –12° C for 24 hours, although the percentage of germination was reduced.

Since conidial germination was not greatly influenced by short periods of freezing, a study of the longevity of conidia held at –12° C was undertaken. A series of hanging drop cultures was prepared and stored at that temperature and at the same time two control cultures were incubated at 2° C to test the viability of the conidia. At intervals of one day or more, duplicate cultures were transferred from the –12° C to the 2° C chamber, where they remained for 24 hours before the percentage of germination was determined. A second series of cultures was later treated in the same manner, except that the slides

were incubated at 4° C after removal from the freezing temperature. The first trial was conducted over a period of 40 days, while the second was extended to cover a period of 100 days.

The results of the trials presented in table 7 show that the percentage of germination is reduced by prolonged freezing. A small amount of germination was observed after the conidia had been stored at –12° C for 40 days, but none occurred after longer periods at the same temperature. From these results it is evident that conidia of *Peronospora schachtii* are not killed by frost and that they could even survive short periods of freezing weather.

TABLE 7

LONGEVITY OF CONIDIA OF PERONOSPORA SCHACHTII FROZEN IN A HANGING DROP AT –12° C

Days at —12° C before germination trials	Per cent germination, trial 1	Per cent germination, trial 2	Days at —12° C before germination trials	Per cent germination, trial 1	Per cent germination, trial 2
1	96	21	10	....	7
2	62	32	15	....	4
3	5	27	20	....	7
4	10	....	25	....	3
5	11	11	30	....	4
6	49	....	40	1	1
7	62	....	50*	....	0
8	0	....	0	91	73
9	0	....	(Control)		

* No conidia germinated after having been frozen for 50 days or more.

The conidia of *Peronospora trifoliorum* de Bary and *Pseudoperonospora humuli* are also able to germinate after short periods of freezing, as has been shown by Melhus and Patel[28] for the former species, and by Arens[4] for the latter.

Infection of host plants by conidia during short periods of favorable conditions depends upon the rapidity of germination and germ tube production. The relation of temperature to the time required for this process was studied by using hanging drop cultures as in previous germination trials. Duplicate cultures were incubated at the same time, and at regular intervals observations were made on the approximate percentage of germination (table 8).

The results presented in table 8 show that, at temperatures from 6° to 25° C, germination was initiated within 2 hours after incubation, while at 4° C, from 3 to 3½ hours were required. At all temperatures the percentage of germination was approximately as great by the end of 4 hours as after 24 hours.

TABLE 8

EFFECT OF TEMPERATURE ON THE TIME REQUIRED FOR CONIDIAL GERMINATION

Temperature, degrees Centigrade	Per cent germination											
	1 hr.	1½ hrs.	2 hrs.	2½ hrs.	3 hrs.	3½ hrs.	4 hrs.	5½ hrs.	7 hrs.	18 hrs.	24 hrs.	48 hrs.
Trial 1:												
4°	0	0	0	0	15	60	90	95	95	95	95	—
6°	0	0	12	50	75	80	95	95	95	95	95	—
8°	0	0	50	75	80	85	95	95	95	95	95	—
Trial 2:												
4°	0	0	0	0	0	40	50	—	60	60	60	60
6°	0	0	0	1	10	20	25	—	25	30	30	30
12°	0	0	3	3	8	9	9	—	10	10	10	10
20°	0	0	1	3	4	4	4	—	4	4	4	5
25°	0	0	1	1	1	2	2	—	2	5	9	28
30°	0	0	0	0	0	0	0	—	0	0	0	0

The relation of temperature to germ tube elongation was determined by measurements made in the same hanging drop cultures which were used for the data presented in table 8. At the specified time intervals the average length of germ tubes in each culture was determined and the results are presented in table 9.

TABLE 9

EFFECT OF TEMPERATURE ON THE TIME REQUIRED FOR GERM TUBE DEVELOPMENT

Temperature, degrees Centigrade	Average length of germ tubes in microns											
	1 hr.	1½ hrs.	2 hrs.	2½ hrs.	3 hrs.	3½ hrs.	4 hrs.	5½ hrs.	7 hrs.	18 hrs.	24 hrs.	48 hrs.
Trial 1:												
4°	0	0	0	0	8	24	32	72	100	240	400	....
6°	0	0	8	16	28	60	94	120	156	320	400	....
8°	0	0	12	24	32	76	104	160	184	320	400	....
Trial 2:												
4°	0	0	0	0	0	3	16	....	60	160	240	320
6°	0	0	0	8	16	20	24	....	80	240	320	400
12°	0	0	16	28	40	62	76	....	170	400	480	580
20°	0	0	24	36	44	56	60	....	150	320	320	340
25°	0	0	48	48	48	48	60	....	120	160	200	320
30°	0	0	0	0	0	0	0	....	0	0	0	0

It is evident that the rate of germ tube growth varied directly with the temperature during the first 3 hours of incubation. However, in trial 1 by the end of 24 hours there was no difference in the average length of germ tubes produced at 4°, 6°, and 8° C. In the second trial

during the first 3 hours the few germ tubes produced at 25° were longer than those produced at lower temperatures. By the end of 24 and 48 hours, however, it was evident that greater germ tube growth occurred at 12°, than above or below this point. Within the first 24 hours germ tubes of 200μ or more were produced at all the temperatures tested, except 30° C.

When conidia on a dry slide were exposed to direct sunlight at 32° to 34° C for 4 hours or more, their power to germinate was destroyed. Those exposed 2 hours or less showed a low percentage of germination after incubation in a drop of water at 4°. Conidia exposed to diffused light indoors at 16° to 25° C for 24 hours or less, did not entirely lose their power to germinate, although the percentage was greatly diminished.

The relation of temperature to the germination of *Peronospora schachtii* conidia was found to be similar to that reported by Hiura[21] for *P. effusa* from *Spinacia oleracea*, except that *Peronospora schachtii* proved to be more tolerant of low temperatures. Hiura found the optimum temperature to be from 8° to 10° C, the minimum below 3°, and the maximum near 30° C. Voglino[41] reported that when conidia of *P. schachtii* were immersed in water or beet decoction for several hours at a temperature of 20–22° C, they germinated by means of zoospores. In the present studies large numbers of conidia have been observed under different conditions and under a range of temperatures from ½° to 30° C, but in no case has a conidium been observed to germinate by means of zoospores.

RELATION OF PARASITE TO HOST TISSUE

Pathogenicity.—To determine if the same conditions which favored conidial germination of *Peronospora schachtii* also favored conidial infection, seedlings of the garden beet variety Detroit Dark Red, growing in steamed soil in four-inch pots, were sprayed with a water suspension of conidia. The inoculum was sprayed on both surfaces of the cotyledons by means of a De Vilbiss atomizer within ten days after the seedlings emerged. Immediately after inoculation the seedlings were incubated for 48 hours at the desired temperature within a chamber in which a nearly saturated atmosphere was maintained by a free water surface. They were then transferred to a greenhouse bench and placed in a chamber kept moist by a layer of wet sphagnum moss. Conidiophores and conidia of *P. schachtii* appeared on the seedlings five to six days after inoculation when the greenhouse

temperature was held between 18° and 24° C. When infected seedlings were stored under similar conditions in a greenhouse held between 8° and 20°, from seven to eight days were required for the development of conidiophores and conidia (table 10). The percentage of infection was determined eight days after the seedlings were inoculated.

TABLE 10

RELATION OF TEMPERATURE TO INFECTION OF BETA VULGARIS SEEDLINGS SPRAYED WITH CONIDIA OF PERONOSPORA SCHACHTII

	Temperature in degrees Centigrade								
	½	2	4	8	10–12	16	20	25	30
Number of trials	4	6	9	8	7	5	7	3	7
Total plants	366	396	728	328	312	631	362	303	333
Number plants infected	314	348	636	296	265	571	282	45	12
Per cent infected	86	88	87	90	85	90	78	15	4

From three to nine trials were conducted at each of nine constant temperatures between ½° and 30° C. The first trial was started on October 15, 1929, and the last on February 10, 1930. The results, summarized in table 10, show that 85 to 90 per cent of the seedlings exposed at temperatures between ½° and 16° C were infected. Seventy-eight per cent of the seedlings were infected at 20°, but at temperatures above that point the percentage was much lower. This information is presented graphically in figure 12. Detailed counts on the percentage of cotyledons and leaves infected were also made but since the results were similar to those for plants they have been omitted from the table. The wide range of temperatures shown to be favorable for infection, as compared to the range favorable for conidial germination, probably may be accounted for by the large quantity of conidia sprayed upon the seedlings.

Judging from the majority of the trials, 8° C is the optimum temperature for conidial infection. The tolerance of this fungus for low temperatures, as indicated in the germination trials, is further shown by the high percentage of infection which occurred at a temperature of ½° C. On one occasion the water in the base of the inoculation chamber was frozen, yet 89 per cent of the plants were infected.

The parts of a beet seedling most susceptible to infection with conidia of *Peronospora schachtii* are the young cotyledons and newly formed leaves. As these structures mature they apparently become

less susceptible. It was found that the cotyledons are susceptible to infection as soon as they emerge from the ground. Whether the relative resistance of leaves of different ages is influenced by the size, number, or distribution of the stomata has not been determined.

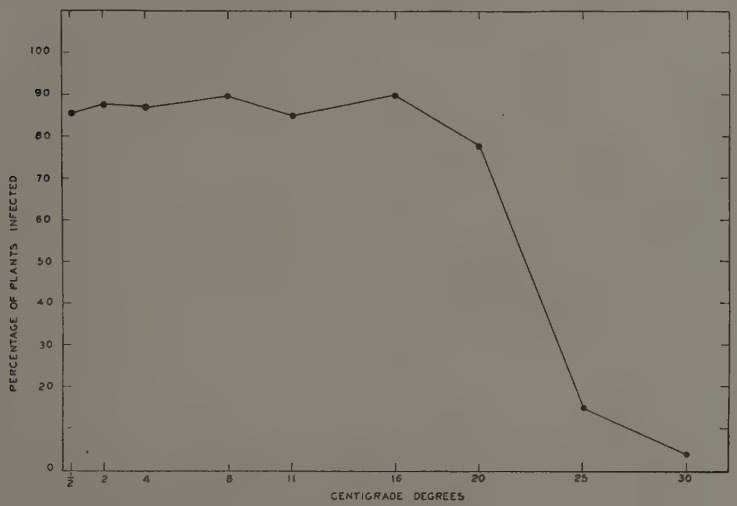

Fig. 12. Relation of temperature to infection of *Beta vulgaris* seedlings by *Peronospora schachtii* conidia.

Mycelial Invasion of the Host.—For observations on the distribution of *Peronospora schachtii* in the tissue of the host, both free-hand and paraffin sections were used. The mycelium and haustoria of *P. schachtii* were, in most cases, readily observed in unstained material, but where the host pigment obscured the fungus mycelium, clearing, by means of 4 per cent potassium hydroxide or concentrated lactic acid, was found advisable. For the study of free-hand sections or macerated tissues, staining with cotton blue in lacto-phenol was found to be more satisfactory than with safranin, although the latter is very useful for quick observations. Magdala red and light green were also used for this purpose. With paraffin sections, Delafield's haemotoxylin and Flemming's triple stain gave the best results. A good differential stain was obtained when sections were left for 48 hours in one part of resorcin blue in 1000 parts of 50 per cent alcohol. Young mycelium and haustoria were stained a brilliant blue, while host material became a dark brown color. The older fungus structures, however, were not so readily distinguished by this stain.

Under field conditions, in the fall of 1929, the first infections were observed in beet root beds six to eight weeks after planting. The fungus was invariably found attacking isolated areas on the older and upper leaves. Attempts to discover a mycelial connection between such infections and the beet crown were made without success. During November, the isolated lesions on the older leaves disappeared or became less conspicuous and the disease appeared abundantly on the young rosette leaves. The new leaves at the center of the rosette were either permeated throughout by the fungus or attacked only along the petiole and base of the blade. Microscopic observations established the fact that there is a continuous mycelial connection between the young infected rosette leaves and the crown of the beet.

It has not been definitely ascertained through what tissues the mycelium gained entrance to the crown, but it seems probable that the infection originated from conidia germinating on the young rosette leaves, rather than from mycelium growing down the petiole from the isolated, diseased areas on the older leaves. Within the crown and neck of the beet, mycelium and haustoria were observed in abundance among all the parenchyma tissues except the pith. In a large number of cases the fungus hyphae were found concentrated near the vascular bundles.

Microscopic studies have shown that mycelium of this fungus may be found throughout the full length of inflorescence axes produced on diseased plants, as was previously reported by Salmon and Ware.[38] The first cauline leaves formed on the floral axis often remain healthy, but the flower shoots produced in the axils of such leaves are usually severely infected. The growth of the fungus keeps pace with the flower development and the mycelium and haustoria can be observed permeating the sepals, pericarp, and filaments, prior to anthesis. Because of the mycelial connection between infected shoots and the crown of the beet through the axis, it is probable that in many cases the infection occurs from the interior of the plant in a manner resembling systemic invasion, rather than from conidial infection on the surface of the flower parts. Following periods of rainfall, however, localized downy mildew infections have often been observed upon the flower parts of otherwise healthy plants. Conidiophores and conidia usually do not appear on the surface of floral parts of *Beta vulgaris* except during cool damp weather. The identity of the fungus mycelium may be definitely established by placing infected floral parts in a moist chamber for a few hours and thus inducing sporulation.

The presence of conidiophores and conidia of *Peronospora arenariae* (Berk.) Tulasne, *P. radii* de Bary, and *P. violacea* Berk. on

the flower parts of host plants has been reported by Fischer.[15] According to Lind,[26] *P. stigmaticola* Raunkiaer occurs only on the flowers of *Mentha aquatica* L. *Peronospora schachtii* was observed on the inflorescence of *Beta vulgaris* by Hollrung[22] and Salmon and Ware[38] but they did not state what tissues were invaded. Recently Cook[11] has shown that mycelium of *Peronospora schleideni* Unger invades the flowers of *Allium cepa* L. and establishes itself within the ovule.[4].

By the use of stained paraffin sections, it has been determined that the mycelium of *Peronospora schachtii* not only permeates the pericarp and sepals of the developing seed ball, but actually invades the integuments of the young ovule. The path of entrance from the pericarp to the integument has not been definitely determined but in some cases mycelial strands were found throughout the full length of the funiculus and oospores were observed near both ends of this structure (fig. 13*C*). The evidence, therefore, points to the funiculus as a possible avenue of entrance into the ovule for the fungus. In many sections of immature seed balls abundant oospores have been observed in the sepals and in the pericarp (fig. 13*A* and *B*).

Material in all stages of development has not been available and, therefore, the relation of the mycelium and oospores in the integument to the developing embryo has not been determined. Penetration of the mycelium into the nucellus and embryo has not been observed. Evidence secured thus far indicates that the fungus mycelium and oospores become imbedded in the seed coat of the mature seed ball. The latter conclusion has been partially verified by dissecting out apparently viable seed from seed balls bearing old conidiophores on their surface. When the seed coat was removed and its inner wall examined microscopically, abundant non-septate mycelium connected to haustoria characteristic of *Peronospora schachtii* was found ramifying among the cells. Similar portions of the seed coats from disease-free seed were entirely free from fungus mycelium.

Although it has not been possible to secure definite proof that the mycelium found within the seed coat is that of *Peronospora schachtii*, the fact that *P. schachtii* conidiophores were found on the surface of the seed balls, that haustoria resembling those of this fungus were

[4] In a recent publication Melhus described and illustrated the presence of mycelium and oospores in the seeds of plants attacked by three species of Peronosporales, *Peronospora alsinearum* Casp. on *Cerastium viscosum* L., *Peronospora viciae* Berk. on *Pisum sativum* L. and *Cystopus bliti* (Biv.) Lév. on *Amaranthus retroflexus* L. (Melhus, I. E. The presence of mycelium and oospores of certain downy mildews in the seeds of their hosts. Iowa State College Jour. Sci. 5:185–188. 1931.)

connected to the mycelium, and that no fungus mycelium was found in apparently disease-free beet seed, makes this conclusion seem probable.

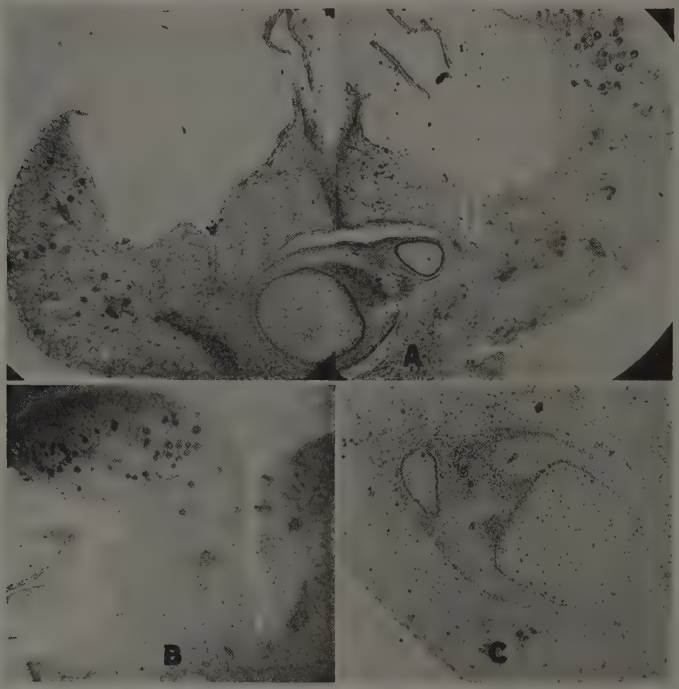

Fig. 13. Oospores of *Peronospora schachtii* in beet seed balls.
A. Longitudinal section of immature seed ball showing oogonia and oospores in the sepals. x44.
B. Oospore production in a sepal. x42.
C. Oospores in the integument of the ovule near the funiculus. x53.

PERENNIAL MYCELIUM OF THE PATHOGENE

Overwintering of several species of the Peronosporaceae by means of perennial mycelium has been established. Melhus[27] compiled the results of his own investigations and those of previous workers, showing that three species of *Phytophthora,* two of *Plasmopara,* one of *Cystopus,* and nine species of *Peronospora* can survive as perennial mycelium.

In addition, Lind[26] states that *Peronospora candida* Fuckel is perennial in the subterranean parts of *Primula elatior* Hill. By means of extensive investigations Murphy,[29] and Murphy and McKay,[30] have shown that *Peronospora schleideni* hibernates as perennial mycelium, while Salmon and Ware[37] and Ware[42] have, in the same way, shown that the mycelium of *Pseudoperonospora humuli* is perennial. Gardner[18] presented evidence that *Peronospora parasitica* (Pers.) de Bary, previously shown by Melhus[27] to survive on *Lepidium virginicum* L., could hibernate in the roots of the turnip (*Brassica rapa* L.). Klebahn[23] has reported that *Peronospora pulveracea* Fuckel lives over as perennial mycelium in the rootstocks of *Helleborus* sp.

Kühn[24] stated that he had proved by repeated experiments that *Peronospora schachtii* can survive the winter as mycelium in the crowns of beets stored for seed production, and reappear on the young leaves when the beets are planted the following spring.

To test the hibernation of the fungus in the crown of stored stecklings, 79 diseased and 18 healthy beets were collected in July, 1929, and stored in sawdust at 4° C for 100 days. The beets were then planted in steamed soil in the greenhouse. Twelve of the healthy plants survived the storage period and none showed any signs of *Peronospora schachtii* on the new leaves. Only 3 of the 79 diseased plants survived and one of these produced leaves bearing typical conidiophores and conidia. The low percentage of plants which survived probably indicates that the conditions of storage were not ideal; but at the same time, a comparison with the healthy plants clearly shows that *P. schachtii* weakened the infected plants and provided a means of entry for saprophytic fungi and bacteria. The one case of successful hibernation, however, indicated that the conclusion of Kühn[24] on this point was justified.

In regions having severe winters the perennial mycelium in stecklings stored for seed production serves as a means of overwintering. In central California the root beds are planted in the early fall, the beet roots or stecklings are transplanted direct to seed fields in December or January, and the entire life cycle of the plant is completed in a ten-month period of continuous growth. Mycelium of *Peronospora schachtii* often invades the crown of beet seedlings while they are still in the root beds and when such plants are moved to the seed fields they initiate centers of infection. The perennial mycelium, in this case, serves to carry the fungus from the root beds to the seed beet fields.

In the same way, the perennial mycelium can carry the fungus through the summer period when weather conditions are unfavorable and when no commercial beet crop is growing in the region devoted to seed production. This was demonstrated in the summer of 1929 on a number of diseased beets which were being maintained in an experimental plot at Davis, California. When extremely hot dry weather occurred, all external symptoms of the disease disappeared from the plants, although microscopic examinations showed that the fungus mycelium was still abundant in the crown of many of the beets. Several months later when the weather was cooler, the conidiophores and conidia of *Peronospora schachtii* appeared on the newly formed leaves. Since there were no other beet plants in the vicinity, this observation serves as a verification of the fact that *P. schachtii* does survive unfavorable periods as mycelium within the beet crown.

The discovery by Prillieux[33] of abundant oospore production by this fungus in beet leaves under certain conditions, provides a second source from which beet plantings may become infected.

Although Prillieux,[33] and Salmon and Ware[38] have suggested the possibility of *Peronospora schachtii* being perpetuated by oospores in the soil, no reference to controlled experiments substantiating this suggestion has been found. Trials have been conducted to secure information as to the possibility of *P. schachtii* being carried over by oospores in the soil. Soil was collected from a seed field and a market garden, both of which had produced diseased crops during the previous season. *Beta vulgaris* seed from a disease-free region was planted in pots filled with the soil collected, part of each lot of soil having been previously steam sterilized for use as a control. Seven hundred and eight beet seedlings developed in 67 pots of field soil, and 153 developed in the 24 pots of sterilized soil. No infection occurred in any of the pots. Because of the meager information available concerning the conditions necessary for oospore germination and infection, the negative data presented above cannot be considered significant.

When seed of *Beta vulgaris* was planted in steam sterilized soil in which had been mixed abundant pulverized leaf material containing oospores of *Peronospora schachtii,* only one seedling in each of two trials became infected. Three other trials gave negative results and no infected seedlings were observed in any of the control pots.

SEED TRANSMISSION

Eriksson[13] in 1924 observed a small number of infected sugar beet plants scattered throughout an otherwise disease-free field and concluded that this condition was probably the result of seed transmission. This hypothesis was given credence by the fact that Hollrung[22] described the presence of *Peronospora schachtii* on the inflorescence of seed beets. The latter author, however, stated that there was no proof that the disease could be transferred through the seed.

Clinton[10] has shown that the mycelium and oospores of *Phytophthora phasaeoli* Thaxt. are present in the seed of lima beans (*Phaseolus lunatus* L.). He found, however, that infected seed which germinated did not produce diseased seedlings and concluded that the primary infection of young seedlings originated not directly from mycelium in the cotyledons, but from zoospores produced by the mycelium or oospores carried over in the seed.

From the results of controlled seed germination trials Angell[1] tentatively concluded that "blue mould" of tobacco (*Peronospora* sp.) can be transmitted by seed from severely infested fields. Cook[11] recently reported the presence of mycelium of *Peronospora schleideni* in the floral parts of *Allium cepa* L. and suggested the possibility of seed transmission.

It has been shown that the mycelium of *Peronospora schachtii* may invade the seed ball and establish itself in the pericarp and inner layers of the seed coat either with or without the formation of oospores (fig. 13*A*, *B* and *C*). The invasion of the nucellus, endosperm, or embryo has not been observed. There is also the possibility that oospores produced elsewhere may become lodged in the crevices of the seed ball and serve as a source of infection to the young seedlings. Unfortunately no method of growing *Peronospora* species in pure culture has yet been devised. Therefore, to determine the viability of the mycelium or oospores within the seed balls, it was necessary to germinate the seed under controlled conditions and observe the presence or absence of seedling infection.

In order to conduct experiments on the seed transmission of this fungus it was necessary to secure a supply of seed known to have been infected, or exposed to infection. A large number of infected flower shoots were tagged soon after pollination. Examination of these shoots at a later date revealed the fact that, almost without

exception, flowers which were infected in an early stage of development failed to mature. Many of the seed balls which developed were so badly shriveled that they could be readily removed by ordinary commercial cleaning practices. Less severely infected flowers did, however, produce viable seed. The material finally selected for the transmission trials consisted of: first, seed from infected plants, second, random selected seed from infested fields, third, commercial seed from diseased fields, and fourth, commercial seed, part of which had been used for planting fields that were found to contain infected plants. Commercial seed from the Pacific Northwest, a region in which *Peronospora schachtii* has never been reported, was used for the controls. The soil used in all trials and the containers were steam sterilized at 40 pounds pressure for two hours. After the seedlings emerged, the flats and pots containing them were enclosed by sterilized cloth covers which were removed only for watering and observation. In the early trials the seedlings were watered with a fine spray to increase the humidity, but in later trials the water was poured down the side of the container to avoid splashing any material on the seedlings.

In the first trial, 14 samples of seed from individual diseased plants were planted on November 15, 1929, and within 60 days one or more infected seedlings appeared in ten of the samples. In addition, one infected seedling was found among 400 produced from random selected seed, while another such seed sample produced none. Diseased seedlings were also found in one of the two lots produced by commercial seed harvested from infested fields and from one of the two lots of seed which had previously produced field plantings of diseased beets. European grown sugar beet seed, which had produced a field planting containing one per cent of infected plants in the spring of 1929, produced three infected seedlings out of 1000.

The second series of trials, started on January 20, 1930, was similar to the first, but on a much larger scale. Two weeks before starting the trials, the benches, walls, and floor of the greenhouse were washed down carefully and all susceptible host material was removed. Steamed soil and containers were used as before and new cloth covers were made.

The temperature was somewhat higher than during the previous trials and apparently more favorable for seedling development, since a shorter period was required for emergence, and subsequent growth was more rapid. That the fungus development was also influenced is shown by the fact that the incubation period from planting to the

appearance of infection was, in this case, from 24 to 33 days, while in the earlier trials from 45 to 60 days had been required.

Out of 21 seed samples from single diseased plants 9 produced infected seedlings, of which 5 had produced diseased seedlings in the former trials. Infected seedlings were also found in three lots of commercial seed harvested from infested fields and in two lots of seeds which had produced fields of infected plants. No infection was observed in five lots of control seedlings.

TABLE 11

RESULTS OF TESTS ON TRANSMISSION OF PERONOSPORA SCHACHTII THROUGH SEED OF BETA VULGARIS COLLECTED FROM INDIVIDUAL DISEASED PLANTS

Sample number	Number trials	Number seedlings	Number seedlings showing primary infection	Per cent primary infection
1	1	200	0	0.00
2	2	1,450	3	0.20
3	2	520	1	0.20
4	2	110	1	0.91
5	2	260	1	0.38
6	2	1,020	6	0.59
7	3	1,720	6	0.35
8	2	740	0	0.00
9	3	1,680	5	0.30
12	1	150	1	0.67
13	3	3,040	10	0.33
14	1	200	1	0.50
16	1	50	0	0.00
20	2	820	1	0.12
23	1	360	0	0.00
24	1	350	1	0.29
25	1	45	0	0.00
26	1	275	0	0.00
29	1	100	0	0.00
30	1	100	0	0.00
35	1	161	0	0.00
36	2	540	1	0.19
40	1	975	1	0.10
15*	1	150	0	0.00
19*	1	400	1	0.25
39*	1	290	0	0.00
Totals for above trials.....	40	15,806	40	0.25

* Composite sample collected from several diseased plants.

The results of all the trials are summarized in tables 11 and 12. Twenty-three seed samples collected from individual diseased plants were planted under isolated conditions, of which 14 have produced infected seedlings in one or more trials. In some cases the same seed

sample produced a few infected seedlings in one trial but none in the second trial. On the other hand, certain samples, such as numbers 7, 9, and 13, produced diseased seedlings in each of the three trials. Irregularities in the results may be accounted for by the apparently small percentage of primary infection. In these studies only the first infected seedlings observed in each container and those showing infection before the elapse of five days, the conidial generation period, were considered to represent primary infections.

TABLE 12

RESULTS OF TESTS ON TRANSMISSION OF PERONOSPORA SCHACHTII THROUGH COMMERCIAL SEED OF GARDEN BEETS, SUGAR BEETS, AND MANGELS

Sample number	Number trials	Number seedlings	Number seedlings showing primary infection	Per cent primary infection
Garden beets				
10	2	1,970	3	0.15
17	3	2,010	5	0.25
21	2	940	1	0.11
22	2	725	1	0.14
61	1	190	0	0.00
62	1	145	0	0.00
63	3	885	0	0.00
64	2	780	0	0.00
65	2	286	0	0.00
66	1	150	0	0.00
69	1	440	0	0.00
70	1	610	0	0.00
71	1	450	1	0.22
72	1	490	1	0.20
73	1	420	0	0.00
74	3	3,490	7	0.20
Sugar beets				
18	4	4,820	8	0.17
Mangels				
38	1	500	0	0.00
Totals for above trials	32	19,301	27	0.14

Results obtained with seed samples 10 and 17, harvested from severely infested commercial seed beet fields, show that not all infected seed balls are removed in cleaning. A few infected seedlings were produced in each planting made from this material. Seed samples 61, 62, and 63 from the Pacific Northwest where *Peronospora schachtii* is as yet unknown, produced no diseased seedlings under similar conditions.

In some cases it has been possible to correlate field observations with greenhouse experiments. For example, seed sample 21, secured through an Eastern seed company but the original source of which is unknown, was used to plant two commercial fields in 1929, both of which later developed infected plants. In one case the beet planting was well isolated, while in the other case it was near another beet planting which remained apparently free of the disease. In the greenhouse trials this seed sample produced a few infected seedlings. Seed sample 18 was used to plant a large commercial sugar beet field in the spring of 1929. In May a count was made in this field and approximately one per cent of the plants, uniformly distributed throughout the planting, were found to be infected. In the greenhouse among 4820 seedlings, 8 primary infections were observed.

The percentage of seedlings which are infected by fungus material carried with the seed ball can only be approximated. From these studies, however, it appears that seed from plants known to be diseased seldom produces more than one per cent of infected seedlings. From the eight samples of commercial seed found to be infected, only 0.18 per cent of the seedlings were diseased. No attempt has been made as yet to determine whether the inoculum carried with the seed ball exists as mycelium or oospores.

Throughout these trials every precaution was taken to prevent outside infection, and since certain samples of seed repeatedly produced infected seedlings, while others always produced seedlings free of infection, the conclusion that *Peronospora schachtii* is transmitted by means of the seed seems justified.

CONTROL

During the progress of these investigations no experimental evidence on the control of *Peronospora schachtii* has been obtained. The information secured points to the advisability of certain practices which should, theoretically, reduce the losses suffered from this disease. Downy mildew of beets is apparently carried with the seed and, therefore, plantings should be made with seed from disease-free sources. The abundant production of oospores under certain conditions no doubt produces soil infestation. No evidence is available as to the longevity of the oospores in the soil, but it is advisable to avoid, wherever possible, planting beets in fields where the disease has previously occurred.

In root beds the presence of downy mildew symptoms on the youngest rosette leaves is usually associated with mycelium in the crown. It therefore seems advisable, when transferring beet plants from the root beds to the seed-beet fields, to eliminate all plants showing this type of infection.

Spraying with Bordeaux mixture and dusting with copper-lime dust are now being practiced by certain growers in an attempt to check the disease in the root beds and seed fields. Although studies are now in progress, no definite information as to the effectiveness of these treatments is yet available.

SUMMARY

Peronospora schachtii has been known to occur to a limited extent on sugar beets in California since 1911 but it was not until 1929 that the disease became a serious factor in garden beet seed production. In 1929 an estimated loss of 43 per cent was incurred from this disease on 600 acres of garden beets grown for seed in the Sacramento Valley. The disease is serious also on market beets in the coastal trucking sections south of San Francisco.

The fungus attacks plants in all stages of development. Under artificial conditions the disease readily appears on cotyledons and young leaves of seedling plants, resulting in a bleaching and downward curling of the cotyledons, and a distortion of the young leaves. Infected cotyledons and young leaves show a heavy gray coating of conidiophores and conidia on their lower surface.

Under field conditions, the first symptoms of downy mildew in the beet root beds usually occur as isolated irregular lesions on older leaves. After the fall rains begin, the disease is confined chiefly to the youngest leaves, causing the center of the beet rosette to become a mass of small, tightly curled and distorted leaves covered with a mass of conidiophores and conidia. In the seed-beet fields, *Peronospora schachtii* infection on the inner rosette leaves is the most conspicuous symptom of the disease, but during periods of rainfall secondary infection occurs as isolated areas on the outer leaves. Flower shoots produced on diseased plants are often invaded systemically, which results in the entire inflorescence showing a stunted and compact form of growth. Infected bracts and flower parts are swollen, distorted, and faded in color.

Field observations on the development of a downy mildew epidemic in a beet root bed during the fall of 1929 showed almost conclusively that all subsequent infection spread from two primary centers.

Inoculation of young plants, under controlled conditions, has shown the following to be susceptible to the disease: *Beta vulgaris* (ten varieties of garden beets, nine varieties of mangels and three varieties of sugar beets), *B. vulgaris* var. *cicla* (two varieties of Swiss chard), *B. bourgaei*, *B. bourgaei* × *B. procumbens*, *B. macrocarpa*, *B. maritima*, *B. patellaris*, *B. patula*, *B. procumbens*, *B. scutellaris*, and *B. vulgaris* var. *abyssinica*. No infection was obtained on *Chenopodium album* and *C. murale*. Experimental evidence showed that the downy mildew on *Beta vulgaris* and that on *Spinacia oleracea* are pathogenically different.

Controlled temperature studies have shown that a temperature of 2°–10° C is favorable for conidial germination, with an optimum between 4° and 7° C. The minimum is below ½°, while the maximum is near 30° C. Conidial germination was initiated within 2 hours at temperatures from 6° to 25° but at 4°, 3 to 3½ hours were required. The length of germ tube varied from 240μ to 480μ at the end of the first 24 hours with the greatest development occuring at 12° C. Short periods of freezing did not affect the viability of conidia and a few were found to be capable of germinating after 40 days at –12° C.

A high percentage of infection was obtained on beet seedlings sprayed with conidial suspensions at temperatures between ½° and 20° C. Slight infection was obtained at 30° C. Cotyledons and newly formed leaves were found to be the most susceptible portions of the beet seedlings.

Abundant oospores were found in the leaves and floral parts of seed beets and in cotyledons and young leaves of seedlings exposed to infection under artificial conditions. Attempts to germinate the oospores have been unsuccessful. Oospore inoculations in sterile soil resulted in infection on a single beet seedling in each of two trials. No infection was obtained in three other trials.

Microscopical studies of free-hand and paraffin sections have shown that the fungus mycelium spreads from the young infected rosette leaves of the beet into the crown. Leaves and flower shoots produced after the mycelium invades the crown are usually completely invaded by the fungus.

Mycelium and oospores were found abundantly in the pericarp and sepals of beet flowers and occasionally were present in the funiculus and the integuments of the ovule. Mycelium and haustoria re-

sembling those of *Peronospora schachtii* were found inside the testa of viable seed from seed balls bearing dry conidiophores on their surfaces.

Twenty-seven samples of beet seed collected from diseased plants and 18 samples of commercial seed were planted in sterilized soil in the greenhouse. Infected seedlings appeared in 15 lots of the former and in 8 lots of the commercial seed. Primary infection occurred on 0.10 to 0.91 per cent of the seedlings from seed produced on diseased plants and on 0.11 to 0.25 per cent of those from commercial seed. Seed from disease-free regions produced no infected seedlings. It was therefore concluded that *Peronospora schachtii* is transmitted with the seed.

Field observations and greenhouse trials confirmed the conclusion of earlier investigators that *Peronospora schachtii* may hibernate by means of perennial mycelium in the beet crown.

No adequate control measures are as yet known. Life history studies and field observations indicate that the use of disease-free seed, avoidance of infested fields, and elimination of infected stecklings are advisable.

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